

# Identification and treatment of eating disorders in people with obsessive–compulsive disorder

*Joanna E Steinglass*

*Department of Psychiatry,  
Columbia University College  
of Physicians & Surgeons,  
Eating Disorders Research  
Unit, New York State  
Psychiatric Institute,  
1051 Riverside Drive,  
New York, NY 10032, USA  
Tel.: +1 212 543 6742;  
Fax: +1 212 543 5607;  
E-mail: js1124@  
columbia.edu*

Patients with obsessive–compulsive disorder (OCD) commonly struggle with an eating disorder as well, which raises several issues for treatment providers. First and foremost, the patient who is seeking treatment for OCD may not have identified or disclosed the presence of an eating disorder, leaving it to the clinician to accurately assess these diagnoses. Diagnosis of an eating disorder in a patient with OCD follows the same guidelines as diagnosis in a patient without OCD. The assessment may be complicated by the patient's lack of insight or ambivalence regarding treatment for the eating disorder (as distinguished from the treatment for OCD). Treatment of OCD can be complicated by the presence of an eating disorder. Patients with anorexia nervosa may not respond to pharmacologic interventions. Malnourishment in patients with anorexia nervosa or bulimia nervosa may impair the patient's participation in psychotherapy for OCD. Clinicians treating patients with OCD need to be adept at diagnosing eating disorders in this population, and will need to consider the role of the eating disorder when planning treatment.

Obsessive–compulsive disorder (OCD) is a common and disabling illness. Reported prevalence rates vary, but lifetime prevalence has been estimated at 2% [1]. The symptoms involve a combination of dysfunctional thoughts and behaviors. The obsessions of OCD are persistent thoughts, impulses or images that are intrusive or unwanted. Compulsions are repetitive, purposeful actions that are usually intended to neutralize or reduce the anxiety raised by the obsessions. Such compulsions are highly ritualized and stereotyped behaviors. The patient has some degree of insight that his/her worries are unrealistic; however, insight in OCD occurs along a spectrum and can be so limited that patients seem delusional [2]. Patients with OCD often have coexisting psychiatric disorders, which can complicate the clinical picture. In particular, there is a high degree of comorbidity with eating disorders [3]. There are phenomenologic similarities between eating disorders and OCD such that the eating disorder diagnosis can be missed in a patient who presents for treatment of OCD. Furthermore, the presence of an eating disorder raises several issues complicating the patient's treatment. This paper aims to provide clinicians treating patients with OCD with a review of the issues that are important in the evaluation and treatment of eating disorders.

Eating disorders encompass a seemingly disparate group of illnesses in terms of behavior and appearance. Anorexia nervosa (AN) is

characterized by a relentless pursuit of thinness and fear of becoming fat, in which patients starve themselves to the extremes of low weight, resulting in amenorrhea and risk of death. Bulimia nervosa (BN) appears somewhat different in that patients are of normal weight but engage in recurrent binge eating and compensatory behaviors, such as vomiting, compulsive exercise or laxative abuse. These behaviors also pose significant health risks; complications of BN can result in a range of morbidities, from relatively minor dental caries to the more significant cardiac arrhythmias and esophageal tears. Binge-eating disorder (BED) is characterized by episodes of excessive intake, with a sense of loss of control and without compensatory behaviors, which frequently results in obesity (although this is not a diagnostic criterion). Collectively, eating disorders are characterized by excessive concerns with shape and weight, and abnormal eating behaviors.

## Comorbidity between OCD & eating disorders

Many studies have suggested a notable degree of comorbidity between OCD and eating disorders [4]. In patients with a primary diagnosis of OCD, lifetime prevalence of an eating disorder has been reported to be 10.2% (3.1% with AN, 3.1% with BN, 1.4% with BED and 3.1% with eating disorder not otherwise specified [EDNOS]) [5]. One study of heritability of

**Keywords:** anorexia nervosa, bulimia nervosa, eating disorder, obsessive–compulsive disorder, treatment

future  
medicine part of fsg

OCD and eating disorders found that OCD spectrum disorders were significantly more common in first-degree relatives of patients with eating disorders (AN and BN) [6].

Rates of OCD in patients with a primary eating disorder vary. Comorbidity with OCD has consistently been reported to be higher in AN than in BN [7,8] yet comorbidity in BN is also more common than in healthy controls [8]. Halmi and colleagues reported that, out of 62 patients with AN, a quarter had a lifetime history of OCD [9]. In a much larger sample of inpatients and outpatients with AN, Godart and colleagues similarly reported a lifetime prevalence of OCD in approximately 25% of patients [10], which is significantly greater than in a matched control population. A review of the literature on comorbidity in AN reported that in patients with AN, the lifetime prevalence of OCD ranged from 10–66%, and prevalence was higher than in a control group [11]. Comorbidity between BED and OCD has not been well studied; however, the one small study in this area suggests that OCD is not more common in patients with BED, and that OCD symptoms in obese patients with BED are comparable to obese non-binge-eating patients and are significantly lower than patients with OCD [12].

While the rate of comorbidity may be over-represented in these studies involving clinical cohorts, the consistent findings of coexistence of these disorders suggests that patients who present with OCD should be assessed for the presence of an eating disorder. While eating disorders are more commonly seen in women, they are not exclusive to women; therefore, heightened awareness of the possibility of an eating disorder is warranted for both sexes. Those who provide treatment for OCD need an understanding of the assessment and treatment of eating disorders.

### Biological overlap between OCD & eating disorders

Several lines of evidence suggest that there may be biological commonalities between OCD and eating disorders. Data from a large study of the genetics of AN and BN have suggested that perfectionism is a personality trait that coaggregates with obsessive-compulsive personality disorder, OCD and eating disorders [13]. Thus, it may be that there is a genetic shared vulnerability to OCD and AN. Cognitive neuroscience research has noted similar deficits on neuropsychological tasks in patients with AN and OCD [14,15]. The

tasks administered in these studies implicate abnormalities in frontostriatal circuits, which are proposed to subserve the pathology of OCD in one prominent model [16,17]. Interestingly, there are case reports of AN triggered by infections (group A  $\beta$ -hemolytic streptococcus, Epstein-Barr and upper respiratory infections), which has been described as ‘autoimmune AN’, similar to reports of postinfectious OCD [18–20]. Disturbances in CNS serotonin function have also been implicated in both disorders [21,22]. These observations provide circumstantial evidence that OCD and AN may share a common neurobiological foundation.

### Making the diagnosis

Both OCD and eating disorders involve a combination of obsessive thoughts and stereotyped behaviors. Patients who present for treatment of OCD need to be asked whether they have any symptoms relating to food, or shape and weight. When obsessions and compulsions are food- or body image-related, the diagnosis of an eating disorder needs to be considered. The presentation of eating disorders can vary and can comprise a range of symptoms, including behavioral, psychological and medical. The evaluation of an eating disorder can be complicated because patients’ descriptions of their symptoms may not directly map onto the diagnostic criteria in the Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV) [23]. For example, a patient is not likely to state that she experiences “overconcern with shape and weight”. Diagnostic assessment is even more complex in the presence of OCD, where eating disorder symptoms may not be spontaneously mentioned by the patient.

In addition, the compromised nutritional status resulting from the eating disorder may bring about worsening of OCD symptoms, thereby masking the primary eating disorder diagnosis. In the starved state, obsessions and compulsions worsen. Thus, underweight patients with AN can appear to have more severe OCD than is the case after weight restoration. Similarly, patients with BN, although normal weight, can be malnourished from their binge-purge behaviors, or from electrolyte imbalances, which can worsen some of their psychological symptoms. Appropriate diagnosis is crucial for treatment; therefore, the clinician needs to be mindful of the common co-occurrence of these disorders and aware of the criteria for the diagnosis of eating disorders.

The most prominent criterion for the diagnosis of AN is a significantly low body weight; the DSM-IV cites less than 85% of ideal body weight as a guideline, though clinical judgment is recommended. DSM-IV criteria also include a fear of gaining weight, a distortion of body image, and amenorrhea for 3 months. BN is characterized by repeated episodes of binge eating followed by a compensatory behavior to avoid weight gain. By definition, patients with BN are of normal weight (patients who binge and purge but are significantly underweight are given the diagnosis of AN, purging subtype). Like in the diagnosis of AN, the diagnosis of BN requires the presence of overconcern with body weight and shape. BED is defined by the presence of recurrent episodes of binge eating (including an excessive amount of food and a sense of loss of control over eating) without compensatory behaviors. Binge episodes are characterized by eating rapidly, feeling uncomfortably full, eating without hunger, and embarrassment or guilt about eating.

When the patient with OCD endorses food- or shape-related concerns, evaluation for an eating disorder needs to be included in the assessment. The components of the evaluation for an eating disorder are similar across all diagnoses (a more detailed discussion of the diagnosis of eating disorders can be found elsewhere [24]). Patients with eating disorders, and particularly patients with AN, may be ambivalent about or uninterested in treatment; therefore, it is important to begin the evaluation with nonjudgmental, open-ended questions, allowing the patient to express feelings about the evaluation. While the therapist must maintain a nonconfrontational stance, asking specific, direct questions is also necessary to get past the patient's ambivalence about disclosing her symptoms. Frequently, this is best done by inquiring about specific eating patterns (frequency and content of all food and drink intake) on a typical day.

Certain typical eating disorder behaviors require specific attention and direct questions. The clinician needs to evaluate the degree of restriction or rules around meals by asking if there are foods that the patient avoids. Assessment of the occurrence and frequency of binge eating is necessary. Binge eating is defined in the DSM-IV as the consumption of an unusually large amount of food, with associated feelings of loss of control. It is important to note that patients with AN may experience small amounts of food as a binge; therefore, the clinician

should ask about the content of a binge. The clinician should inquire about occurrence and frequency of purging behaviors. These behaviors include self-induced vomiting and laxative or diuretic abuse. Less commonly, patients may chew and spit out food without swallowing. In patients with diabetes mellitus, purging can also occur as insulin omission. The clinician should also assess the frequency and intensity of exercise and the patient's commitment to exercise (i.e., will they forgo other activities in order to continue exercising).

Weight throughout the patient's lifetime (highest and lowest weight) and salient weight changes are also important in clarifying the possible history of eating disorders other than the current diagnosis. In female patients, menstrual history is important. AN is characterized by 3 months of amenorrhea, but BN can also present with amenorrhea. Absence of menses presents a significant risk to the patient, including risk of osteoporosis.

Any assessment of a patient with an eating disorder needs to include a clinician's measurement of height and weight. The clinician's measurement is necessary, as people are known to provide inaccurate estimations of height and weight, irrespective of the presence of an eating disorder. Further medical evaluation should be recommended, including pulse and blood pressure, general medical exam, and laboratory tests (a review of the medical complications of eating disorders can be found elsewhere [25]).

### Considerations in the diagnosis of AN

Patients' with AN may demonstrate denial of symptoms and denial of severity of the problem, which complicates the evaluation. As with OCD [26], insight in AN has been described as occurring along a spectrum, with a subgroup of patients demonstrating delusional levels of conviction in their disordered beliefs [27]. Patients who have identified OCD as their problem may well minimize their food- and shape-related beliefs and rituals, thereby masking a primary diagnosis of an eating disorder. Furthermore, it is common for all patients with AN to deny a fear of gaining weight in interview, so this diagnostic criterion must be inferred from the patient's behavior (i.e., inability to maintain a normal weight).

Importantly, the patient may be unaware of the impact of low weight on their obsessions and compulsions. The association between starvation and emergence of psychological

symptoms (e.g., food-related obsessions and rituals, depression and anxiety) has been well documented [28]. Weight gain is known to lead to improvement in these symptoms in patients with AN [29,30]. The clinician may need to provide psychoeducation to the patient, explaining that the underweight state exacerbates obsessions and compulsions. Some patients have nonfood-related obsessions and compulsions, which may resolve, or significantly improve, with weight restoration. For this reason, the diagnosis of OCD may need to be reassessed after the patient has achieved normal weight.

### Treatment

The current mainstays of treatment of eating disorders overlap with the current treatments of OCD: psychological interventions, including cognitive therapy, behavioral therapy and psychopharmacologic interventions, such as selective serotonin-reuptake inhibitors (SSRIs). However, treatment of eating disorders can also include family therapy and nutritional counseling. Treating a patient with OCD and a concurrent eating disorder requires initial determination of the patient's nutritional status. A patient who is nutritionally compromised presents two major concerns in the treatment of OCD: the eating disorder, and the level of malnutrition, may be exacerbating OCD symptoms; and the patient may be too nutritionally compromised to benefit from OCD treatment. In the first scenario, treatment of the eating disorder may ameliorate the OCD symptoms such that treatment recommendations with respect to OCD may change. In the second scenario, the patient's response to treatment may be impeded owing to cognitive impairment, which limits participation in cognitive-behavioral psychotherapy, or may be biologically altered such that medications prove ineffective [29].

Presence of both OCD and an eating disorder may have implications for the course of each illness. Although the data are not entirely consistent [31], comorbid OCD has been associated with a longer duration of AN and BN [32]. This suggests that the presence of OCD may have an impact on the patient's prognosis with respect to the eating disorder.

### *Anorexia nervosa*

SSRIs are a mainstay of the treatment of OCD. Owing to the phenomenologic similarities and the overlap between these disorders, SSRIs were originally thought to be promising for the

treatment of AN. However, in a randomized, controlled trial, fluoxetine provided no additional benefit compared with placebo with respect to weight gain in inpatients with AN. In addition, while both groups showed significant improvement in psychological measures (depression, anxiety or body image), there was no difference between medication and placebo [29]. Furthermore, a large trial of weight-restored outpatients with AN found that fluoxetine provided no additional benefit compared with placebo with respect to relapse prevention in outpatients receiving cognitive-behavioral therapy (CBT) for relapse prevention [33]. Therefore, there is cause for concern that nutritional status may compromise psychopharmacologic treatment of OCD.

Given the disappointing results of pharmacologic interventions across medication classes (antidepressants, antipsychotics or mood stabilizers) for patients with AN, treatment recommendations rely on psychological interventions. For underweight patients, these include participation in inpatient or day programs aimed at weight normalization. After weight normalization, emerging evidence supports the utility of CBT. One study demonstrated that CBT was more effective than standard treatment (nutritional counseling) for relapse prevention [34]. CBT for AN differs somewhat from CBT for OCD in that interventions focus on cognitive distortions and do not include formal exposure therapy and response prevention. Many studies have reported cognitive deficits in underweight patients, especially related to attention [35]. Therefore, there is cause for concern that patients would have difficulty participating in psychotherapy for OCD, as therapy requires attentional capacity as well as learning, which may be compromised at low weight.

Treatment for AN is in need of further developments, as current treatments continue to yield low response rates and high relapse rates [36]. Nutritional rehabilitation is a critical first step in the treatment of all patients with AN [37], and is especially important when considering treatment for coexisting OCD. As has been emphasized throughout this review, OCD symptoms improve with weight gain alone [30]. For severely underweight patients, structured settings such as inpatient or partial hospital may be required in order to achieve normal weight. In these settings, behaviorally based treatments have been very useful in normalizing weight. Pharmacologic treatments for weight gain in

AN have been disappointing. While anecdotal reports of successful treatments have been published, only a small number of randomized, controlled trials have been conducted. For a detailed review, see Steinglass and Walsh [38]. All randomized, controlled trials have indicated that medications are not helpful in underweight patients with AN, with respect to mood and anxiety symptoms in addition to eating disorder pathology. The absence of definitive medication treatment highlights a need for improved psychological interventions. There are notable similarities between AN and OCD [39]; therefore, treatment for AN may be improved by drawing from current understanding of treatment for OCD and placing increased emphasis on behavioral techniques.

#### *Bulimia nervosa*

Contrary to the treatment of AN, pharmacological interventions have been useful in the treatment of BN. Several randomized, controlled trials have demonstrated the benefits of SSRIs in patients with BN [38]. One large study demonstrated that fluoxetine 60 mg is of greater benefit than fluoxetine 20 mg [40], suggesting that, similar to the treatment of OCD, higher doses are of benefit in this population. This may simplify the pharmacologic management of comorbid OCD and BN. In addition to antidepressants, topiramate has also been shown to be useful [41,42].

The data on the use of antidepressants in the treatment of BN are convincing in indicating that fluoxetine is safe and beneficial. While it is likely that other SSRIs would be effective, only fluoxetine has been examined in placebo-controlled trials, and should be used in a dose of 60 mg/day. Most patients can be rapidly titrated to this dose over the course of a week. Bupropion is not recommended in the treatment of BN because of the risk of seizure [43]. There are consistent indications that medications modestly enhance the benefits of psychological treatment. Psychotherapy trials have demonstrated that CBT for BN is effective in reducing binge eating and vomiting episodes, and is the psychotherapy of choice [44]. Studies that have combined CBT with antidepressant medication suggest that there may be an advantage to combined treatment over medication alone [45].

Patients with BN are, by definition, of normal weight. Nonetheless, their disordered eating patterns may compromise cognitive function [46].

One commonly accepted model suggests that binge eating is a response to restraint and resulting hunger. Patients with BN may exhibit restraint outside of binge episodes such that they are experiencing effects of starvation, although not to the same extent as patients with AN. In addition, binge eating and purging behavior can lead to electrolyte disturbances, which may compromise cognitive function. Some studies have demonstrated cognitive impairments in patients with BN, especially with respect to impulsivity [47]. Thus, in patients with significant illness, it may be important to see improvement in binge-purge behavior before embarking on psychological treatment for OCD.

#### *Binge eating disorder*

Treatment of BED in a patient with OCD is less complicated than the other eating disorders, as treatment strategies are not in conflict. Several medication strategies have shown promise in the treatment of BED, including antidepressants and weight-loss medications [48,49]. SSRIs are generally considered first-line treatment for binge eating because of their favorable side-effect profile, although they do not demonstrate an effect on weight loss. In addition, topiramate has demonstrated efficacy in decreasing frequency of binge episodes and in decreasing weight [48]. Topiramate can be combined with SSRIs. Sibutramine has also been shown to be helpful for BED [50], although combining sibutramine with other serotonergic antidepressants poses a theoretical increased risk of serotonin syndrome. Psychotherapy, including individual and group CBT, has also been shown to be useful in the treatment of BED [51].

#### **Conclusion & future perspective**

When treating patients with OCD, clinicians need to be alert to the possible presence of an eating disorder for several reasons. First, the high degree of comorbidity between illnesses indicates that the clinician evaluating a patient with OCD should specifically inquire, in detail, about symptoms of an eating disorder. Second, the presence of AN or BN may complicate the treatment of OCD such that the eating disorder would need to be addressed and some improvement seen prior to embarking on treatment of the OCD. For patients with AN, weight restoration may be necessary for psychopharmacologic and psychotherapeutic interventions to be helpful. Third, the clinician should be aware that



coexistence of OCD with an eating disorder may worsen the patient's prognosis for recovery from the eating disorder, although study results vary.

There are phenomenologic similarities between obsessions and compulsions in OCD and food- and shape-related obsessions and rituals in eating disorders, which suggests a shared pathophysiology between these disorders. There may be neurobiologic features in these disorders that represent a shared vulnerability. Patients may have a neurologic predisposition toward obsessive, intrusive ideation and stereotyped behavioral responses that puts them at risk for either disorder. Brain imaging research in patients with eating disorders is needed to evaluate frontostriatal circuits in order to determine

whether these disorders share similar neural mechanisms with OCD. Treatment innovations are particularly needed in AN. Given the disappointing results from pharmacologic interventions, new treatments may be improved by capitalizing on the utility of techniques used in CBT for OCD that have not been formally studied in some eating disorder populations, such as development of exposure therapy and ritual prevention for patients with AN. Translating from clinical research in anxiety disorders has potential to improve our understanding of the mechanism of eating disorders, as well as identify new pharmacologic approaches, such as those that are beginning to show promise in enhancing learning in anxiety disorders [52].

### Executive summary

- Patients with obsessive-compulsive disorder (OCD) have high rates of comorbid eating disorders, including anorexia nervosa and bulimia nervosa.
- The presence of an eating disorder can be masked by the patient's reluctance to spontaneously report symptoms; therefore, the clinician needs to actively inquire about specific details regarding eating behavior.
- Starvation and malnutrition that occurs with both anorexia nervosa and bulimia nervosa can lead to worsening of obsessions and compulsions in OCD.
- Treatment needs to prioritize normalization of eating behavior, and particularly weight, in order to best treat the patient's OCD.

### Bibliography

1. Fontenelle LF, Mendlowicz MV, Versiani M: The descriptive epidemiology of obsessive-compulsive disorder. *Prog. Neuropsychopharmacol. Biol. Psychiatry* 30(3), 327–337 (2006).
2. Kozak M, Foa EB: Obsessions, overvalued ideas, and delusions in obsessive-compulsive disorder. *Behav. Res. Ther.* 32, 343–353 (1994).
3. Kaye WH, Bulik CM, Thornton L, Barbarich N, Masters K; the Price Foundation Collaborative Group: Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am. J. Psychiatry* 161(12), 2215–2221 (2004).
4. Hsu GLK, Kaye W, Weltzin T: Are the eating disorders related to obsessive compulsive disorder? *Int. J. Eat. Disord.* 14(3), 305–318 (1993).
5. Pinto A, Mancebo MC, Eisen JL, Pagano ME, Rasmussen SA: The Brown Longitudinal Obsessive Compulsive Study: clinical features and symptoms of the sample at intake. *J. Clin. Psychiatry* 67(5), 703–711 (2006).
6. Bellodi L, Cavallini MC, Bertelli S, Chiapparino D, Riboldi C, Smeraldi E: Morbidity risk for obsessive-compulsive spectrum disorders in first-degree relatives of patients with eating disorders. *Am. J. Psychiatry* 158(4), 563–569 (2001).
7. Godart N, Berthoz S, Rein Z *et al.*: Does the frequency of anxiety and depressive disorders differ between diagnostic subtypes of anorexia nervosa and bulimia? *Int. J. Eat. Disord.* 39(8), 772–778 (2006).
8. Lilienfeld LR, Kaye WH, Greeno CG *et al.*: A controlled family study of anorexia nervosa and bulimia nervosa: psychiatric disorders in first-degree relatives and effects of proband comorbidity. *Arch. Gen. Psychiatry* 55(7), 603–610 (1998).
9. Halmi KA, Eckert E, Marchi P, Sampugnaro V, Apple R, Cohen J: Comorbidity of psychiatric diagnoses in anorexia nervosa. *Arch. Gen. Psychiatry* 48, 712–718 (1991).
10. Godart NT, Flament MF, Curt F *et al.*: Anxiety disorders in subjects seeking treatment for eating disorders: a DSM-IV controlled study. *Psychiatry Res.* 117(3), 245–258 (2003).
11. Godart NT, Flament MF, Perdereau F, Jeammet P: Comorbidity between eating disorders and anxiety disorders: a review. *Int. J. Eat. Disord.* 32(3), 253–270 (2002).
12. Fontenelle LF, Mendlowicz MV, de Menezes GB *et al.*: Comparison of symptom profiles of obese binge eaters, obese non-binge eaters, and patients with obsessive-compulsive disorder. *J. Nerv. Ment. Dis.* 190(9), 643–646 (2002).
13. Halmi KA, Tozzi F, Thornton LM *et al.*: The relation among perfectionism, obsessive-compulsive personality disorder and obsessive-compulsive disorder in individuals with eating disorders. *Int. J. Eat. Disord.* 38(4), 371–374 (2005).
14. Steinglass JE, Stern Y, Walsh BT: Set shifting deficit in anorexia nervosa. *J. Int. Neuropsychol. Soc.* 12(3), 431–435 (2006).
15. Sherman BJ, Savage CR, Eddy KT *et al.*: Strategic memory in adults with anorexia nervosa: are there similarities to obsessive compulsive spectrum disorders? *Int. J. Eat. Disord.* 39(6), 468–476 (2006).

16. Rauch SL, Whalen PJ, Dougherty D, Jenike MA: Neurobiological models of obsessive compulsive disorders. In: *Obsessive Compulsive Disorders: Practical Management*. Jenike MA, Baer L, Minichiello WE (Eds). Mosby, MA, USA 222–253 (1998).
17. Graybiel AM, Rauch SL: Toward a neurobiology of obsessive–compulsive disorder. *Neuron* 28, 343–347 (2000).
18. Sokol MS: Infection-triggered anorexia nervosa in children: clinical description of four cases. *J. Child Adolesc. Psychopharmacol.* 10(2), 133–145 (2000).
19. Sokol MS, Gray NS: Case study: an infection-triggered, autoimmune subtype of anorexia nervosa. *J. Am. Acad. Child Adolesc. Psychiatry* 36(8), 1128–1133 (1997).
20. Henry MC, Perlmutter SJ, Swedo SE: Anorexia, OCD, and streptococcus. *J. Am. Acad. Child Adolesc. Psychiatry* 38(3), 228–229 (1999).
21. Kaye WH, Gendall K, Strober M: Serotonin neural function and selective serotonin reuptake inhibitor treatment in anorexia and bulimia nervosa. *Biol. Psychiatry* 44, 825–835 (1998).
22. Blier P, de Montigny C: Possible serotonergic mechanisms underlying the antidepressant and anti-obsessive–compulsive disorder responses. *Biol. Psychiatry* 44(5), 313–323 (1998).
23. Association AP: *Diagnostic and Statistical Manual of Mental Disorders (4th Edition)*. APA, Washington, DC, USA (1994).
24. Walsh BT, Satir DA: Diagnostic Issues. In: *Assessment of Eating Disorders*. Mitchell J, Peterson CB (Eds). Guilford Press, NY, USA 1–16 (2005).
25. Walsh BT: Eating Disorders. In: *Harrison's Principles of Internal Medicine (16th Edition)*. Kasper DL, Braunwald E, Fauci AS, Hauser SL, Longo DL, Jameson JL (Eds). McGraw Hill, NY, USA 430–433 (2005).
26. Eisen JL, Phillips KA, Coles ME, Rasmussen SA: Insight in obsessive compulsive disorder and body dysmorphic disorder. *Compr. Psychiatry* 45(1), 10–15 (2004).
27. Steinglass JE, Eisen JL, Attia E, Mayer L, Walsh BT: Is anorexia nervosa a delusional disorder? An assessment of eating beliefs in anorexia nervosa. *J. Psychiatr. Pract.* 13(2), 65–71 (2007).
28. Keys A, Brozek J, Henschel A, Mickelsen O, Taylor HL: *The Biology of Human Starvation*. University of Minnesota Press, MN, USA (1950).
29. Attia E, Haiman C, Walsh BT, Flater SR: Does fluoxetine augment the inpatient treatment of anorexia nervosa? *Am. J. Psychiatry* 155(4), 548–551 (1998).
30. Pollice C, Kaye WH, Greeno CG, Weltzin TE: Relationship of depression, anxiety, and obsessiveness to state of illness in anorexia nervosa. *Int. J. Eat. Disord.* 21(4), 367–376 (1997).
31. Thiel A, Zuger M, Jacoby GE, Schussler G: Thirty-month outcome in patients with anorexia or bulimia nervosa and concomitant obsessive–compulsive disorder. *Am. J. Psychiatry* 155(2), 244–249 (1998).
32. Milos G, Spindler A, Ruggiero G, Klaghofer R, Schnyder U: Comorbidity of obsessive–compulsive disorders and duration of eating disorders. *Int. J. Eat. Disord.* 31(3), 284–289 (2002).
33. Walsh BT, Kaplan AS, Attia E *et al.*: Fluoxetine after weight restoration in anorexia nervosa: a randomized controlled trial. *JAMA* 295(22), 2605–2612 (2006).
34. Pike KM, Walsh BT, Vitousek K, Wilson GT, Bauer J: Cognitive behavior therapy in the posthospitalization treatment of anorexia nervosa. *Am. J. Psychiatry* 160(11), 2046–2049 (2003).
35. Green MW, Elliman NA, Wakeling A, Rogers PJ: Cognitive functioning, weight change and therapy in anorexia nervosa. *J. Psychiatr. Res.* 30(5), 401–410 (1996).
36. Steinhausen HC: The outcome of anorexia nervosa in the 20th century. *Am. J. Psychiatry* 159(8), 1284–1293 (2002).
37. Association AP: Treatment of patients with eating disorders, 3rd edition. *Am. J. Psychiatry* 163(Suppl. 7), 4–54 (2006).
38. Steinglass JE, Walsh BT: Medication treatment in anorexia nervosa, bulimia nervosa and binge eating disorder. In: *Eating Disorders*. Brewerton TD (Ed.). Marcel Dekker, NY, USA (2004).
39. Steinglass JE, Walsh BT: Habit learning and anorexia nervosa: a cognitive neuroscience hypothesis. *Int. J. Eat. Disord.* 39(4), 267–275 (2006).
40. Fluoxetine in the treatment of bulimia nervosa. A multicenter, placebo-controlled, double-blind trial. Fluoxetine Bulimia Nervosa Collaborative Study Group. *Arch. Gen. Psychiatry* 49(2), 139–147 (1992).
41. Hoopes SP, Reimherr FW, Hedges DW *et al.*: Treatment of bulimia nervosa with topiramate in a randomized, double-blind, placebo-controlled trial, part I: improvement in binge and purge measures. *J. Clin. Psychiatry* 64(11), 1335–1341 (2003).
42. Nickel C, Tritt K, Muehlbacher M *et al.*: Topiramate treatment in bulimia nervosa patients: a randomized, double-blind, placebo-controlled trial. *Int. J. Eat. Disord.* 38(4), 295–300 (2005).
43. Horne RL, Ferguson JM, Pope HG Jr *et al.*: Treatment of bulimia with bupropion: a multicenter controlled trial. *J. Clin. Psychiatry* 49(7), 262–266 (1988).
44. Wilson GT: Cognitive behavior therapy for eating disorders: progress and problems. *Behav. Res. Ther.* 37(Suppl. 1), S79–S95 (1999).
45. Agras WS, Rossiter EM, Arnow B *et al.*: Pharmacologic and cognitive–behavioral treatment for bulimia nervosa: a controlled comparison. *Am. J. Psychiatry* 149(1), 82–87 (1992).
46. Woodside BD, Staab R: Management of psychiatric comorbidity in anorexia nervosa and bulimia nervosa. *CNS Drugs* 20(8), 655–663 (2006).
47. Duchesne M, Mattos P, Fontenelle LF, Veiga H, Rizo L, Appolinario JC: Neuropsychology of eating disorders: a systematic review of the literature. *Rev. Bras. Psiquiatr.* 26(2), 107–117 (2004).
48. McElroy SL, Hudson JL, Capece JA, Beyers K, Fisher AC, Rosenthal NR: Topiramate for the treatment of binge eating disorder associated with obesity: a placebo-controlled study. *Biol. Psychiatry* 61(9), 1039–1048 (2007).
49. Milano W, Siano C, Putrella C, Capasso A: Treatment of bulimia nervosa with fluvoxamine: a randomized controlled trial. *Adv. Ther.* 22(3), 278–283 (2005).
50. Appolinario JC, Bacaltchuk J, Sichiari R *et al.*: A randomized, double-blind, placebo-controlled study of sibutramine in the treatment of binge-eating disorder. *Arch. Gen. Psychiatry* 60(11), 1109–1116 (2003).
51. Munsch S, Biedert E, Meyer A *et al.*: A randomized comparison of cognitive behavioral therapy and behavioral weight loss treatment for overweight individuals with binge eating disorder. *Int. J. Eat. Disord.* 40(2), 102–113 (2006).
52. Anderson KC, Insel TR: The promise of extinction research for the prevention and treatment of anxiety disorders. *Biol. Psychiatry* 60(4), 319–321 (2006).